Amendments to the Claims:

This listing of claims will replace all prior versions, and listings, of claims in the instant application:

Listing of Claims:

Claims 1-37. (Cancelled)

Claim 38. (Previously Presented): A method for determining the susceptibility of an at least partially atrophied thymus to reactivation in a patient, comprising:

monitoring the level in the patient's blood or serum of a marker associated with activation of the thymus;

disrupting sex steroid-mediated signaling to the thymus of the patient;

monitoring the level in the patient's blood or serum of the marker; and

comparing the level of the marker before and after disruption of sex steroid-mediated signaling,

wherein an early increase in the level of the marker following disruption of sex steroid-mediated signaling indicates susceptibility of the patient's thymus to reactivation.

Claim 39. (Previously Presented): The method of claim 38, wherein the patient has a disease that at least in part atrophied the thymus of the patient.

Claim 40. (Previously Presented): The method of claim 38, wherein the patient has had a treatment of a disease, wherein the treatment at least in part atrophied the thymus of the patient.

Claim 41. (Previously Presented): The method of claim 40, wherein the treatment is immunosuppression, chemotherapy, or radiation treatment.

Claim 42. (Previously Presented): The method of claim 38, wherein the patient is post-pubertal.

Claim 43. (Previously Presented): The method of claim 38, wherein the sex steroid-mediated signaling to the thymus is disrupted by surgical castration.

Claim 44. (Previously Presented): The method of claim 38, wherein the sex steroid-mediated signaling to the thymus is disrupted by chemical castration.

Claim 45. (Previously Presented): The method of claim 38, wherein the sex steroid-mediated signaling to the thymus is disrupted by administration of a pharmaceutical.

Claim 46. (Previously Presented): The method of claim 45, wherein the pharmaceutical is selected from the group consisting of LHRH agonists, LHRH antagonists, anti-LHRH vaccines, anti-androgens, anti-estrogens, SERMs, SARMs, SPRMs, ERDs, aromatase inhibitors, anti-progestogens, Dioxalan derivatives, and combinations thereof.

Claim 47. (Previously Presented): The method of claim 46, wherein the LHRH agonists are selected from the group consisting of Goserelin, Leuprolide, Lupron, Triptorelin, Meterelin, Buserelin, Histrelin, Nafarelin, Lutrelin, Leuprorelin, Deslorelin, Cystorelin, Decapeptyl, Gonadorelin, and combinations thereof.

Claim 48. (Previously Presented): The method of claim 46, wherein the LHRH antagonists are selected from the group consisting of Abarelix, Cetrorelix, and combinations thereof.

Claim 49. (Previously Presented): The method of claim 46, wherein the pharmaceutical is a combination of a LHRH agonist and a LHRH antagonist.

Claim 50. (Previously Presented): The method of claim 38, wherein the early increase occurs within four weeks following disruption of sex steroid-mediated signaling.

Claim 51. (Previously Presented): The method of claim 38, wherein the early increase occurs within two weeks following disruption of sex steroid-mediated signaling.

Claim 52. (Previously Presented): The method of claim 38, wherein the early increase occurs within one week following disruption of sex steroid-mediated signaling.

Claim 53. (Previously Presented): The method of claim 38, wherein the early increase occurs within about 4 to 5 days following disruption of sex steroid-mediated signaling.

Claim 54. (Previously Presented): The method of claim 38, wherein the early increase occurs within about 2 to 3 days following disruption of sex steroid-mediated signaling.

Claim 55. (Previously Presented): The method of claim 38, wherein the early increase occurs within about 24 hours following disruption of sex steroid-mediated signaling.

Claim 56. (Previously Presented): The method of claim 38, wherein the marker is a thymopoietic hormone or thymopoietic cytokine.

Claim 57. (Previously Presented): The method of claim 56, wherein the marker is selected from the group consisting of IL-7, Factor Thymique Serique (FTS), thymulin, thymosin, thymosin-alpha 1, thymosin-beta 4, thymopoietin, CXCL12, CCL19, CCL21, CCL22, CCL25, a member of the keratinocyte growth factor (KGF) family, a member of the fibroblast growth factor (FGF) family and any combination thereof.

Claims 58-60. (Cancelled)

Claim 61. (Previously Presented): A method for determining the susceptibility of an at least partially atrophied thymus to reactivation in a patient, comprising:

monitoring the *in vitro* proliferative responsiveness of T cells in the patient's blood;

disrupting sex steroid-mediated signaling to the thymus of the patient;

monitoring the *in vitro* proliferative responsiveness of the T cells in the patient's blood; and

comparing the *in vitro* proliferative responsiveness of the T cells in the patient's blood before and after disruption of sex steroid-mediated signaling to the thymus of the patient,

wherein an early increase in the *in vitro* proliferative responsiveness of the T cells following disruption of sex steroid-mediated signaling indicates susceptibility of the patient's thymus to reactivation.

Claim 62. (Previously Presented): A method for determining the susceptibility of an at least partially atrophied thymus to reactivation in a patient, comprising:

monitoring the level of newly produced T cells in the patient's blood;

disrupting sex steroid-mediated signaling to the thymus of the patient;

monitoring the level of newly produced T cells in the patient's blood; and

comparing the level of the newly produced T cells in the patient's blood before and after disruption of sex steroid-mediated signaling,

wherein an early increase in the level of the newly produced T cells following disruption of sex steroid-mediated signaling indicates susceptibility of the patient's thymus to reactivation.

Claim 63. (Previously Presented): The method of claim 62, wherein the monitoring of the level of newly produced T cells is accomplished by monitoring a marker selected from the group consisting of Ki67, CD62L, CD45RA, CD69, LFA-1, VCAM, ICAM-1, VLA-4 and any combinations thereof.

Claim 64. (Previously Presented): The method of claim 62, wherein the monitoring of the level of newly produced T cells is accomplished by monitoring T Cell Receptor Excision Circles (TRECs).

Claim 65. (Cancelled):

Claim 66. (Previously Presented): The method of claim 64, wherein the TREC levels are monitored by a method comprising:

purifying the patient's T cells;

isolating DNA from the purified T cells; and

performing real-time polymerase chain reaction on the isolated DNA with TREC-specific primers and a molecular beacon,

wherein the primers amplify the TREC DNA, and wherein the molecular beacon detects the amplified TREC DNA.

Claim 67. (Previously Presented): The method of claim 66, wherein the TREC-specific primers are selected from the group consisting of SEQ ID NO:1, SEQ ID. NO:2, SEQ ID NO:3, and SEQ ID NO:4.

Claim 68. (Previously Presented): The method of claim 64, wherein the patient has a disease that at least in part atrophied the thymus of the patient.

Claim 69. (Previously Presented): The method of claim 64, wherein the patient has had a treatment of a disease, wherein the treatment at least in part atrophied the thymus of the patient.

Claim 70. (Previously Presented): The method of claim 69, wherein the treatment is immunosuppression, chemotherapy, or radiation treatment.

Claim 71. (Previously Presented): The method of claim 64, wherein the patient is post-pubertal.

Claim 72. (Previously Presented): The method of claim 64, wherein the sex steroid-mediated signaling to the thymus is disrupted by surgical castration.

Claim 73. (Previously Presented): The method of claim 64, wherein the sex steroid-mediated signaling to the thymus is disrupted by chemical castration.

Claim 74. (Previously Presented): The method of claim 64, wherein the sex steroid-mediated signaling to the thymus is disrupted by administration of a pharmaceutical.

Claim 75. (Previously Presented): The method of claim 74, wherein the pharmaceutical is selected from the group consisting of LHRH agonists, LHRH antagonists, anti-LHRH vaccines, anti-androgens, anti-estrogens, SERMs, SARMs, SPRMs, ERDs, aromatase inhibitors, anti-progestogens, Dioxalan derivatives, and combinations thereof.

Claim 76. (Previously Presented): The method of claim 75, wherein the LHRH agonists are selected from the group consisting of Goserelin, Leuprolide, Lupron, Triptorelin, Meterelin, Buserelin, Histrelin, Nafarelin, Lutrelin, Leuprorelin, Deslorelin, Cystorelin, Decapeptyl, Gonadorelin, and combinations thereof.

Claim 77. (Previously Presented): The method of claim 75, wherein the LHRH antagonists are selected from the group consisting of Abarelix, Cetrorelix, and combinations thereof.

Claim 78. (Currently Amended): The method of claim 74, wherein [[the]] the pharmaceutical is a combination of a LHRH agonist and a LHRH antagonist.

Claim 79. (Previously Presented): The method of claim 65, wherein the early increase occurs within four weeks following disruption of sex steroid-mediated signaling.

Claim 80. (Previously Presented): The method of claim 65, wherein the early increase occurs within two weeks following disruption of sex steroid-mediated signaling.

Claim 81. (Previously Presented): The method of claim 65, wherein the early increase occurs within one week following disruption of sex steroid-mediated signaling.

Claim 82. (Previously Presented): The method of claim 65, wherein the early increase occurs within about 4 to 5 days following disruption of sex steroid-mediated signaling.

Claim 83. (Previously Presented): The method of claim 65, wherein the early increase occurs within about 2 to 3 days following disruption of sex steroid-mediated signaling.

Claim 84. (Previously Presented): The method of claim 65, wherein the early increase occurs within about 24 hours following disruption of sex steroid-mediated signaling.

Claims 85-88. (Cancelled)

Claim 89. (Previously Presented): A method for determining the susceptibility of an at least partially atrophied thymus to reactivation in a patient, comprising:

disrupting sex steroid-mediated signaling to the thymus of the patient; and

monitoring the level in the patient's blood or serum of a marker associated with activation of the thymus,

wherein an early increase in the level of the marker following disruption of sex steroid-mediated signaling indicates susceptibility of the patient's thymus to reactivation.

Claim 90. (Previously Presented): A method for determining the susceptibility of an at least partially atrophied thymus to reactivation in a patient, comprising:

disrupting sex steroid-mediated signaling to the thymus of the patient; and

monitoring the *in vitro* proliferative responsiveness of the T cells in the patient's blood,

wherein an early increase in the *in vitro* proliferative responsiveness of the T cells following disruption of sex steroid-mediated signaling indicates susceptibility of the patient's thymus to reactivation.

Claim 91. (Cancelled)

Claim 92. (Previously Presented): A method for determining the susceptibility of an at least partially atrophied thymus to reactivation in a patient, comprising:

disrupting sex steroid-mediated signaling to the thymus of the patient; and

monitoring the level of newly produced T cells in the patient's blood,

wherein an early increase in the level of the newly produced T cells following disruption of sex steroid-mediated signaling indicates susceptibility of the patient's thymus to reactivation.

Claim 93. (Cancelled):

Claim 94. (Previously Presented): A method for determining the susceptibility of an at least partially atrophied thymus to reactivation in a patient, comprising:

disrupting sex steroid-mediated signaling to the thymus of the patient; and

monitoring the intracellular cytokine levels in the T cells in the patient's blood,

wherein an early increase in the intracellular cytokine levels in the T cells following disruption of sex steroid-mediated signaling indicates susceptibility of the patient's thymus to reactivation.

Claim 95. (Previously Presented): A method for determining the susceptibility of an at least partially atrophied thymus to reactivation in a patient, comprising:

monitoring the intracellular cytokine levels in the T cells in the patient's blood;

disrupting sex steroid-mediated signaling to the thymus of the patient;

monitoring the intracellular cytokine levels in the T cells in the patient's blood; and

comparing the intracellular cytokine levels in the T cells in the patient's blood before and after disruption of sex steroid-mediated signaling,

wherein an early increase in the intracellular cytokine levels in the T cells following disruption of sex steroid-mediated signaling indicates susceptibility of the patient's thymus to reactivation.

Claim 96. (Previously Presented): The method of claim 61, wherein the *in vitro* proliferative responsiveness of T cells in the patient's blood is determined by monitoring proliferation of T cells after anti-CD3 crosslinking.

Claim 97. (Previously Presented): The method of claim 90, wherein the *in vitro* proliferative responsiveness of T cells in the patient's blood is determined by monitoring proliferation of T cells after anti-CD3 crosslinking.

Claim 98. (Previously Presented): The method of claim 92, wherein the level of newly produced T cells in the patient's blood is determined by monitoring the level of the TRECs in the patient's blood.

Claim 99. (Previously Presented): The method of claim 38, wherein the sex steroid-mediated signaling to the thymus is disrupted by lowering the level of sex steroid hormones.

Claim 100. (Previously Presented): The method of claim 46 or 75, wherein the anti-androgen is Eulexin or ketoconazole.